THE IMPORTANCE OF CUTANEOUS SILENT PERIOD IN UREMIC POLYNEUROPATHY

Seyit Mehmet Kayacan Assoc. Prof. MD,[1] Barış İşak MD,[2] Dilek Kayacan MD,[3] Muharrem Müftüoğlu MD,[1] Aytaç Karadağ MD[1]

[1] İstanbul University, İstanbul Medical Faculty, Department of Internal Medicine, İstanbul

[2] Marmara University, Medical Faculty, Department of Neurology, İstanbul

[3] Haydarpaşa Numune Education and Research Hospital, Department of Neurology, İstanbul

ABSTRACT

Objective: *Cutaneous silent period (CSP) is a brief pause in a voluntary contraction following strong stimulation of a cutaneous nerve. Clinical interest in the CSP stems from its potential usefulness for evaluating segment and components of sensory nerves that are not well assessed by standard electro diagnostic methods. Aim of this study was to determine whether CSP has precedence over standard nerve conduction studies (SNCS) in uremic polyneuropathy.*

Material and Method: *The study included 20 chronic hemodialysis patients. Mean age was 42.6 (22-63). Mean uremic period before dialysis and dialysis age were 13.9+7.2 (6-36) and 51.2+46.8 months (12-190), respectively. The eight of patients were symptomatic (paresthesia, pain-burning), the others were asymptomatic. Twenty healthy volunteers were accepted as control group. Standard sensorial and motor nerve conductions and F- responses were recorded in patients. Cutaneous silent period were measured from abductor pollicis brevis (APB) muscle by stimulating third finger and from tibialis anterior muscle by stimulating sural nerve.*

Results: *The significant difference between dialysis and control groups was not found in SNCS. But, at the recording from APB muscle, CSP latency (ms) (82.44+7.80) in hemodialysis patients was longer than controls (63.60+13.70) ($p<0.001$) and CSP duration (ms) (42.10+9.95) was significantly shorter than controls (51.54+11.24) ($p<0.001$). At the recording from tibialis anterior muscle, CSP latency (101.90+13.38) was similar with controls (95.03+12.59) ($0.1<p<0.5$) and CSP duration (34.12+16.09) was significantly shorter than controls (52.95+18.13) ($p<0.001$).*

Conclusion: *The shortening of CSP duration was thought an antecedent and important method for uremic polyneuropathy as thin fiber neuropathy.*

Key Words: *Cutaneous silent period, polyneuropathy, uremia.* ***Nobel Med 2011; 7(3): 89-94***

ÜREMİK POLİNÖROPATİDE KUTANÖZ SESSİZ PERİYODUN ÖNEMİ

ÖZET

Amaç: Kutanöz sessiz periyod (KSP), istemli kasılma esnasında bir kutanöz sinire uygulanan şiddetli bir stimulasyonun ardından kısa süreli duraklamadır. Kutanöz sessiz periyodun klinik önemi, standart elektrodiagnostik yöntemlerle çok iyi anlaşılamayan duysal sinirlerin segment ve bileşenlerinin analizinde yararlı olmasından kaynaklanır. Çalışmanın amacı, üremik polinöropatide KSP'nin standart sinir ileti çalışmalarına erken tanıda üstünlüğü olup olmadığının ortaya konulmasıdır.

Materyal ve Metod: Çalışmaya hemodiyaliz tedavisi gören 20 kronik böbrek yetersizliği hastası alındı. Ortalama yaş 42,6 (22-63) idi. Ortalama diyaliz öncesi üremik dönem ve diyalize giriş süresi sırasıyla 13,9+7,2 ay (6-36) ve 51,2+46,8 ay idi (12-190). Hastaların 8'i semptomatik (uyuşma, yanma hissi), 12'si asemptomatikti. Kontrol grubu, 20 sağlıklı gönüllüden oluştu. Her hastada standart duysal ve motor sinir iletileri ve F-yanıtları kaydedildi. Üçüncü parmak, halka elektrodu ile uyarılarak abduktor pollisis brevis (APB) ve sural sinir uyarımına cevaben tibialis anterior kasından KSP bakıldı.

Bulgular: Standart sinir ileti incelemeleri açısından diyaliz ve kontrol grubu arasında anlamlı bir fark bulunamadı. Ama APB kasından kayıtlı KSP latansı (ms) hemodiyaliz hastalarında (82,44+7,80) kontrollerden (63,60+13,70) daha uzundu ($p<0,001$). Hemodiyaliz hastalarındaki KSP süresi (ms) (42,10+9,95) anlamlı olarak kontrollerden (51,54+11,24) daha kısaydı ($p<0,001$). Tibialis anterior kasından kayıtlı KSP distal latansı (101,90+13,38) kontrollerdekine (95,03+12,59) benzerdi ($0,1<p<0,5$). KSP süresi (34,12+16,09) anlamlı olarak kontrollerden (52,95+18,13) daha kısaydı ($p<0.001$).

Sonuç: Standart sinir ileti incelemelerinin normal olduğu diyaliz hastalarında KSP süresindeki azalmanın varlığı, bu bulgunun ince lif nöropatisinin baskın olduğu üremik polinöropati için öncül ve önemli bir parametre olduğunu düşündürmüştür.

Anahtar Kelimeler: Kutanöz sessiz periyod, polinöropati, üremi. **Nobel Med 2011; 7(3): 89-94**

INTRODUCTION

Peripheral neuropathy in patients with end stage renal disease (ESRD) typically presents as distal symmetrical polyneuropathy, which affects lower extremities greater than upper extremities. The condition exhibits an early asymptomatic stage, and it progresses over months into symptomatic stage. Men are affected more frequently than women. It usually shows up when glomerular filtration rate drops down below 12 milliliter per minute. The most frequently encountered clinical symptoms emerge as a consequence of involvement of large fibers. Involvement of large diameter nerve fibers (Aα), thin myelinated fibers (Aδ) and unmyelinated fibers cause paresthesias, reduction in deep tendon reflexes, impaired vibration sense, weakness, and muscle wasting. The prevalence of this condition varies from 60% to 100%.[1]

During the cutaneous silent period (CSP) temporary suppression of muscle contraction is achieved through postsynaptic inhibition of motor neurons or presynaptic inhibition of the excitatory inputs to motorneurons that sustain voluntary contraction. Cutaneous afferent neurons link up on a variety levels of nervous system; so they have influence upon motor activity. Stimulation of motor nerves during produces a mixture of excitatory and inhibitory effects, which depend on the site and intensity of input, the specific muscle involved and whether the task requires contraction or relaxation of muscle. Cutaneous reflexes are useful and rewarding to study the mechanisms for sensorimotor integration at spinal and supraspinal levels. One of the most powerful cutaneous reflexes is the cutaneous silent period, which is a short period of pause in voluntary contraction following robust stimulation of a cutaneous nerve (Figure 1).[2] Silent period is transient suppression of electrical activity of a muscle during maximum effort, following supramaximal stimulation of its nerve. The principle hypotheses of the electrically generation of cutaneous silent period comprise of spindle afferents, cutaneous afferents, inhibitory spinal reflex system by internuncial neurons and active inhibition descending from the motor cortex.[3-5] According to various reports, CSP is a combination of temporary cessation of muscle spindle discharge, Golgi tendon and cutaneous afferents.[6] It is thought that cutaneous silent period is generated by thin myelinated, high-threshold cutaneous afferents with slow conduction velocity. Cutaneous silent period is an inhibitory spinal reflex mediated by Aδ cutaneous afferents.[7,8] There are →

also findings supporting this theory. High intensity stimulation, typically 10 times the perceptual threshold, is necessary to evoke the cutaneous silent period. Strong stimulation of a cutaneous nerve is followed by synchronized CSP in several muscle groups. Characteristic distribution between upper and lower limb, and cranial muscles depends on the site of stimulation. Clinical importance of cutaneous silent period is that it provides beneficial information for evaluating segments and components of sensory nerves that are not well assessed by current electrodiagnostic methods.[9] In addition, it provides information to understand central nervous system disorders that impair motor and sensory processing.
The aim of our study was to determine the role of cutaneous silent period in early diagnosis of uremic neuropathy.

MATERIAL and METHOD

Our study included 20 chronic renal failure patients who were on dialysis program during time of the study conducted. Of 20 patients, 13 were males, 7 were females with a mean age of 42.6 (22-63) years. The underlying cause of end stage renal failure was chronic glomerulonephritis in 10 out of 20 patients.

The primary renal diseases were unknown in the remaining of the group. Eight of patient had related symptoms (numbness and burning sensation). The mean body mass index was found to be 25.65±2.67 (18.42-29.68) kg/m^2. The mean duration of dialysis was 51.2±46.8 months (12-190). Dialysis adequacy measured by Kt/V was achieved in all cases [Kt/V: 1.68±0.08 (1.48-1.78)]. The control group was consisted of age- and gender-matced, 20 healthy subjects, with no history of trauma to upper or lower limb, or systemic or neurological diseases resulting in peripheral neuropathy. Mean body mass index in control group was 26.69±4.45 (19.92-35.0). No significant statistical difference in height, weight and body mass index was observed between two groups. Patients had hereditary neuropathy, diabetes mellitus, vasculitis, connective tissue disorders, porphyria, primary or secondary amyloidosis, chronic liver disease, history of alcohol intake, thyroid dysfunction and infections (HIV etc.). None of the patients in this study were using drugs for tuberculosis or malignancy. Patients with vitamin B_{12} levels less than 200 mcg/dl were also excluded from the study. Informed consent was obtained from all patients and healthy subjects.

The study was approved by Ethics Committee of Institute of Health Sciences of Marmara University. Electrophysiological studies were performed with

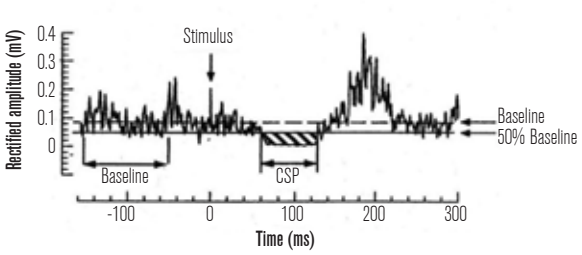

Figure 1. Example of measurements from an average of 10 rectified EMG traces. The baseline (dashed lines) was defined as the average EMG during a 100-ms period prior to the stimulus. The area of the CSP (shading) can be used as a measure of the completeness of EMG suppression.

Medelec Synergy 5 Channel EMG-EP device. Unilateral median, ulnar, radial, bilateral sural, superficial peroneal, medial plantar, dorsal sural sensory; unilateral ulnar, median motor; bilateral peroneal, tibial motor conduction studies, unilateral ulnar, median, bilateral tibial and peroneal F- wave studies were conducted. CSPs were obtained from thenar muscles and tibialias anterior muscle on stimulating digit III with ring electrode and sural nerve respectively. Cutaneous silent period was assessed with the patients and healthy subjects in the supine position and they were instructed to contract tibialis anterior and abductor pollicis brevis muscles with submaximal effort (80%). Sweep time was set at 200-250 ms and sensitivity at 100-200 µV. Sural nerve was stimulated below the lateral malleolus with wave pulses of 0.5 ms and 25 mA intensity. Ten successive responses were recorded and stimuli were applied at a maximum frequency of 1 Hz. Measurement of subsequent 10 silent periods was performed. Ten responses obtained from subjects were superimposed on an ossiloscope. Absolute silent period was measured and preliminary CSP latencies and durations for patients and healthy control group were determined. Median F-responses were recorded from abductor pollicis brevis by the supramaximal stimulus of median nerve at the wrist. Abductor digiti minimi was employed to record ulnar F-responses. Ulnar nerve was stimulated supramaximally at the wrist. Tibial F-responses were recorded from abductor hallusis brevis by supramaximal stimulation of tibial nerve at the medial malleoulus. Peroneal F-responses were recorded from extensor digitorum brevis by supramaximal stimulation of peroneal nerve. Ten subsequent responses were recorded and the shortest latency response was used for calculations. Paired t-test was used for statistical analysis.

RESULTS

The means and statistical comparisons of electromyography measures [distal latency (ms),→

Table 1: The mean+SD and statistically evaluations of EMG findings in dialysis and control groups.

	Dialysis group	Control group	p		Dialysis group	Control group	p
LMP distal latency	2.51+0.46	2.10+0.50	p>0.5	**RPT F latency**	50.52+5.59	45.59+4.54	p<0.5
LMP amplitude	8.37+4.32	9.48+4.45	p>0.5	**LP distal latency**	4.53+0.87	3.65+0.72	p<0.5
LMP velocity	51.01+8.79	56.57+8.17	p>0.5	**LP amplitude**	4.29+1.40	4.19+1.15	p>0.5
RMP distal latency	2.52+0.38	2.06+0.32	p<0.5	**LP velocity**	45.12+5.18	51.10+3.86	p<0.5
RMP amplitude	7.73+3.45	9.83+4.62	p>0.5	**LP F latency**	47.28+5.66	42.64+4.83	p<0.5
RMP velocity	51.71+6.22	54.41+9.70	p>0.5	**RP distal latency**	4.18+0.69	3.65+0.60	p>0.5
LDS distal latency	2.40+0.43	1.92+0.46	p<0.5	**RP amplitude**	5.15+1.77	4.69+0.99	p>0.5
LDS amplitude	6.81+3.59	9.11+5.74	p>0.5	**RP velocity**	44.76+4.94	49.21+3.62	p<0.5
LDS velocity	40.99+6.98	43.78+7.26	p>0.5	**RPF latency**	47.61+7.86	43.16+3.91	p>0.5
RDS distal latency	2.39+0.40	2.07+0.49	p>0.5	**RSM distal latency**	2.50+0.40	2.41+0.31	p>0.5
RDS amplitude	6.77+2.94	8.54+5.60	p>0.5	**RSM amplitude**	35.78+11.80	45.42+14.91	p>0.5
RDS velocity	41.16+5.98	43.31+9.01	p>0.5	**RSM velocity**	52.85+11.99	57.67+6.19	p>0.5
LSP distal latency	2.18+0.37	1.97+0.32	p>0.5	**RSU distal latency**	2.32+0.31	2.12+0.28	p>0.5
LSP amplitude	12.21+6.36	13.63+4.06	p>0.5	**RSU amplitude**	29.82+11.40	38.67+15.31	p>0.5
LSP velocity	52.13+8.36	53.12+6.92	p>0.5	**RSU velocity**	52.46+4.66	58.23+5.80	p<0.5
RSP distal latency	2.16+0.44	2.08+0.21	p>0.5	**RSR distal latency**	1.96+0.33	1.62+0.25	p<0.5
RSP amplitude	13.75+7.54	14.20+5.66	p>0.5	**RSR amplitude**	24.04+8.60	30.87+10.29	p>0.5
RSP velocity	51.50+8.77	52.29+7.53	p>0.5	**RSR velocity**	57.12+4.90	64.64+6.61	p<0.5
LS distal latency	2.40+0.38	2.29+0.42	p>0.5	**RMM distal latency**	3.25+0.32	3.00+0.38	p>0.5
LS amplitude	18.57+5.79	17.05+6.27	p>0.5	**RMM amplitude**	9.63+1.94	9.35+1.84	p>0.5
LS velocity	50.06+6.30	55.17+7.76	p>0.5	**RMM velocity**	56.77+5.48	60.35+5.50	p>0.5
RS distal latency	2.34+0.42	2.43+0.33	p>0.5	**RMM F latency**	25.68+2.18	24.30+1.98	p>0.5
RS amplitude	20.11+7.32	18.88+7.44	p>0.5	**RMU distal latency**	2.59+0.37	2.26+0.34	p<0.5
RS velocity	50.30+5.15	51.86+5.80	p>0.5	**RMU amplitude**	10.32+1.91	10.54+1.64	p>0.5
LPT distal latency	4.73+1.08	4.01+0.74	p>0.5	**RMU velocity**	58.99+6.45	64.23+7.12	p>0.5
LPT amplitude	10.01+2.91	9.52+2.76	p>0.5	**RMU F latency**	26.71+2.16	24.85+2.37	p>0.5
LPT velocity	44.33+6.09	45.55+3.13	p>0.5	**CSP-T1 (APB)**	82.44+7.80	63.60+13.70	p<0.001
LPT F latency	48.33+7.31	45.63+4.90	p>0.5	**CSP-D (APB)**	42.10+9.95	51.54+11.24	p<0.001
RPT distal latency	4.84+0.78	4.03+0.80	p<0.5	**CSP-T1 (TA)**	101.90+13.38	95.03+12.59	0.1<p<0.5
RPT amplitude	9.65+2.42	9.54+2.54	p>0.5	**CSP-D (TA)**	34.12+16.09	52.95+18.13	p<0.001
RPT velocity	44.93+4.97	46.09+4.07	p>0.5				

LMP: Left medial plantar, RMP: Right medial plantar, LDS: Left dorsal sural, RDS: Right dorsal sural, LSP: Left superficial peroneal RSP: Right superficial peroneal, LS: Left sural, RS: Right sural, LPT: Left posterior tibial, RPT: Right posterior tibial, LP: Left peroneal, RP: Right peroneal,, RSM: Right sensory median, RSU: Right sensory ulnar, RSR: Right sensory radial, RMM: Right motor median, RMU: Right motor ulnar, CSP: Cutaneous silent period, APB: Abductor pollicis brevis, TA: Tibialis anterior

amplitude (mV) and conduction velocity (m/s)] and cutaneous silent period measures [T1 (latancy, ms) and D (duration, ms)] belong to dialysis and control group is displayed on the Table 1.

In the standard nerve conduction studies, we found no difference between the disease and control group in terms of left medial plantar (distal latency, amplitude and velocity) and right medial plantar (amplitude and velocity) measures (p>0.5). Similarly, we found no difference among right dorsal sural, left superficial peroneal, right superficial peroneal, left sural, right sural and left posterior tibial measures (distal latency, amplitude and velocity) (p>0.5). Distal latencies of left dorsal sural and right posterior tibial nerves, together with F latency of right posterior tibial nerve found different (p<0.5). There was no difference between the amplitude and velocity of conduction in the left dorsal sural and right posterior tibial nerves (p>0.5).

Only differences observed between two groups were distal latency, velocity and F latency in the left peroneal nerve; velocity in the right peroneal nerve; velocity in the right sensory ulnar nerve; distal latency and velocity in the right sensory radial nerve, distal latency in the right motor ulnar nerve (p<0.5). There were no difference in other measures recorded in these nerves (p>0.5).

CSP durations of patients recorded in both abductor pollicis brevis (42.10±9.95, 51.54±11.24) and tibialis anterior (34.12±16.09, 52.95±18.13) was significantly lower than the control group (p<0.001).

CSP distal latency of patients recorded in lower extremity (101.90±13.38) was not significantly different than the control group (95.03±12.59) (0.1<p<0.5). CSP distal latency of patients recorded in upper extremity (82.44±7.80) was significantly longer than the control group (63.60±13.70) (p<0.001). →

DISCUSSION

Cutaneous silent period is a relative or an absolute diminished activity in electromyography of a voluntarily contracting muscle observed after supra-maximal electrical stimulation of a cutaneous nerve. Afferents of this reflex are composed of A-delta class; they are slow conducting, thinly myelinated nerves conveying painful stimuli. It has been thought that this reflex occurs via shared interneurons inhibiting spinal motor neurons; and among these interneurons Renshaw cells comes forward. Many studies have been conducted about the place and significance of cutaneous silent period in uremic polineuropathy. In one of these studies, electromyographic investigation carried out on peripheral nerves of 42 dialysis patients (30 hemodialysis, 12 chronic peritoneal dialysis patients). All of the standard nerve conduction investigations carried out in polineuropathy has been found abnormal.[10] In general we found no statistical difference between the patient and control group in our study ($p>0.5$).

Another study involved 20 hemodialysis patients. While a significant decrease in CSP duration observed in short-term dialysis patients (a mean dialysis age of 31 months), this difference between patient and control group disappeared in patients undergoing longer dialysis durations (a mean dialysis age of 51 months).[11] This gave rise to the thought that the decrease in CSP duration is an early finding of hemodialysis during the progression of uremic polineuropathy. However, our study demonstrated that after starting hemodialysis, CSP durations recorded from abductor pollicis brevis (42.10 ± 9.95, 51.54 ± 11.24) and tibialis anterior (34.12 ± 16.09, 52.95 ± 18.13) significantly decrease regardless of time, whether it is a short or long term dialysis (a dialysis age of 12 months at least, 190 months at most, 51.2 months on average) ($p<0.001$). Therefore, it can be inferred that CSP duration is a noteworthy parameter in uremic polineuropathy and it is associated with thin fiber neuropathy.

A CSP study carried out in diabetes mellitus demonstrated that CSP distal latency can be affected parallel with standard nerve conduction tests or with thick fiber neuropathies; it has been postulated that CSP duration is associated with thin fiber neuropathy. Prolongation of CSP distal latency is interpreted as thick fiber involvement.[12] While the study on diabetics only covers CSP distal latency and duration recorded on abductor pollicis brevis, our study covers CSP distal latency and duration recorded on tibialis anterior and the former. Quantitative sensory testing applications in end-stage renal patients expose high perception thresholds; this is more obvious in lower extremities.

Lack of difference between CSP distal latencies in the lower extremities of patients (101.90 ± 13.38) and the control group (95.03 ± 12.59) is explained with the lack of a significant difference between the heights of subjects in each group ($0.1<p<0.5$). On the other hand, significant prolongation of CSP distal latencies in the upper extremities of patients (82.44 ± 7.80) in contrast with control group (63.60 ± 13.70) is explained with a slight predisposition to carpal tunnel syndrome due to a probable beta-2 microglobulin deposition related amyloid accumulation in the arm without fistula in uremic patients; it can be revealed by way of sensitive methods.[13]

Even though there is no significant difference between dialysis group and the control group in terms of standard nerve conduction studies, a significant decrease observed in CSP duration ($p<0.001$). This gave rise to the thought that the decrease in the duration of CSP is a more preferential and important parameter for thin fiber neuropathy predominant uremic polineuropathy.

CONCLUSION

While previous studies have reported a disturbance in standard nerve conduction studies and CSP duration in uremic patients, standard nerve conduction studies were not very different than the control in this study but CSP durations were significantly low. Thus, the decrease in the duration of CSP is a preferential and important parameter for uremic polineuropathy.

Previous studies revealed an association between CSP duration and dialysis duration in such a way that CSP duration lowers when shorter time spent during dialysis but when longer time spent CSP duration of patients equalizes with the control group. This study demonstrated that even though the time spent during dialysis is long, CSP duration appears to be short with respect to the control group.

It is statistically disclosed that there were no significant CSP latency difference in the lower extremities of patients and the control group ($0.1<p<0.5$). This can be explained with the lack of a significant difference between the heights of subjects in each group and with the absence of thick fiber involvement; and this gave rise to the thought that a decrease in CSP duration in uremic patients favors thin fiber involvement.

CORRESPONDING AUTHOR: Seyit Mehmet Kayacan Assoc. Prof. MD. Göztepe Çiftehavuzlar Tepegöz sokak Beylerbeyi Apt. 2/11 Kadıköy, İstanbul kayacansm@yahoo.com

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REFERENCES

1. Krishnan AV, Kiernan MC. Uremic neuropathy: Clinical features and new pathophysiological insights. Muscle Nerve 2007; 35: 273-290.
2. Shahani BT, Young RR. Studies of the normal human silent period. Ed: Desmedt JE, New developments in electromyography and clinical neurophysiology. Karger, Basel, 1973: 589-602.
3. Leis AA, Kofler M, Ross MA. The silent period in pure sensory neuropathy. Muscle Nerve 1992; 15: 1345-1348.
4. McLellan DL. The electromyographic silent period produced by supramaximal electrical stimulation in normal man. J Eurol Neurosurg Psychiat 1973; 36: 334-341.
5. Cantello R, Gianelli M, Civardi C, Mutani R. Magnetic brain stimulation: the silent period after the motor evoked potential. Neurology 1992; 42: 1951-1959.
6. Metron PA. The silent period in a muscle of the human hand. J Physiol 1951; 114: 183-198.
7. Cruccu G, Agostino R, Inghilleri M, et al. Mandibular nerve involvement in diabetic polyneuropathy and chronic inflammatory demyelinating polyneuropathy. Muscle Nerve 1998; 21: 1673-1679.
8. Ertekin C. Sentral ve periferik EMG (Anatomi-Fizyoloji-Klinik), Bölüm 19: Refleksoloji ve geç yanıtlar. Meta Basım Matbaacılık Hizmetleri, İzmir 2006: 777-846.
9. Floeter MK. Cutaneous silent periods. Muscle Nerve 2003; 28: 391-401.
10. Tilki HE, Akpolat T, Coşkun M, Stalberg E. Clinical and electrophysiologic findings in dialysis patients. J Electromyogr Kinesiol 2007; 1-9.
11. Resende LAL, Caramori JC, Kimaid PAT, Martin LC, Barretti P. The silent period in patients with chronic renal failure undergoing hemodialysis. Electromyogr Clin Neurophysiol 2002; 42: 275-279.
12. Yaman M, Uludüz D, Yüksel Ş, Pay G, Kızıltan ME. The cutaneous silent period in diabetes mellitus. Neurosci Lett 2007; 419: 258-262.
13. Svilpauskaite J, Truffert A, Vaiciene N, Magistris MR. Cutaneous silent periods in carpal tunnel syndrome. Muscle Nerve 2006; 33: 487-493.